AMENDMENTS TO THE CLAIMS

Please replace all previously pending claims with the following listing of claims:

1. **(Currently amended)** A method for treating a disorder in which TNFα activity is detrimental comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose ~~therapy~~ of 0.01 – 0.1 mg/kg, such that the disorder is treated.

2. **(Original)** The method of claim 1, wherein the disorder is arthritis.

3. **(Original)** The method of claim 2, wherein the disorder is rheumatoid arthritis.

4. **(Currently amended)** The method of any one of claims 2 ~~and~~ or 3, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

5. **(Previously presented)** The method of any one of claims 1-3, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

6. **(Previously presented)** The method of claim 5, wherein the TNFα fusion protein is etanercept.

7. **(Canceled)**

8. **(Currently amended)** A low dose method to alleviate symptoms associated with a disorder in which TNFα activity is detrimental, comprising administering a low dose of 0.01 – 0.1 mg/kg of a TNFα inhibitor to a subject suffering from said disorder, such that the symptoms are treated.

9. **(Original)** The method of claim 8, wherein the disorder is arthritis.

10. **(Original)** The method of claim 9, wherein the disorder is rheumatoid arthritis.

11. **(Currently amended)** The method of any one of claims 9 ~~and~~ or 10, wherein symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

12. **(Previously presented)** The method of any one of claims 8-10, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

13. **(Previously presented)** The method of claim 12, wherein the TNFα fusion protein is etanercept.

14. **(Canceled)**

15. **(Currently amended)** A method for treating arthritis comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose ~~therapy~~ of 0.01 – 0.1 mg/kg, such that the arthritis is treated.

16. **(Original)** The method of claim 15, wherein the arthritis is rheumatoid arthritis.

17. **(Currently amended)** The method of ~~either~~ any one of claims ~~claim~~ 15 or 16, wherein arthritis is treated by alleviating symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

18 **(Currently amended)** The method of any one of claims ~~claim~~ 15 or 16 wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

19. **(Previously presented)** The method of claim 18 wherein the TNFα fusion protein is etanercept.

20. **(Canceled)**

21. **(Currently amended)** A low dose method for treating symptoms associated with arthritis comprising administering to a subject ~~a low dose of~~ an effective amount of a TNFα inhibitor in a low dose of 0.01 – 0.1 mg/kg, such that the symptoms are alleviated.

22. **(Previously presented)** The method of claim 21, wherein the arthritis is rheumatoid arthritis.

23. **(Currently amended)** The method of any one of claims 21 ~~and~~ or 22, wherein the symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

24. **(Original)** The method of claim 23, wherein the symptoms are further selected from the group consisting of joint distortion, swelling, joint deformation, ankylosis on felxion, and severely impaired movement.

25. **(Currently amended)** The method of any one of claims 21 or 22, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

26. **(Previously presented)** The method of claim 25, wherein the TNFα inhibitor fusion protein is etanercept.

27. **(Canceled)**

28. **(Currently Amended)** A method of sequestering TNFα into complexes in a subject suffering from a disorder in which TNFα activity is detrimental, by administering a low dose of 0.01 – 0.1 mg/kg of a TNFα inhibitor to the subject.

29. **(Original)** The method of claim 28, wherein the serum level of TNFα is higher than the serum level of TNFα in a subject not suffering from a disorder in which TNFα activity is detrimental.

30. **(Previously presented)** The method of claim 28, wherein the anti-TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

31. **(Previously presented)** The method of any one of claims 1, 8, or 15, wherein the TNFα inhibitor is administered with an additional therapeutic agent.

32. **(Previously presented)** The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

33. **(Previously presented)** The method of claim 12, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

34. **(Previously presented)** The method of claim 18, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

35. **(Previously presented)** The method of claim 25, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

36. **(Previously presented)** The method of claim 30, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

37. **(Previously presented)** The method of claim 30, wherein the TNFα fusion protein is etanercept.

38. **(Canceled)**

39. **(Canceled)**

40. **(Previously presented)** The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is human.

41. **(Previously presented)** The method of claim 40, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less.

42. **(Currently amended)** A low dose method for treating rheumatoid arthritis in which TNFα activity is detrimental comprising administering to a subject a low dose of 0.01 – 0.1 mg/kg of a human TNFα antibody, or an antigen-binding portion thereof, such that the disorder is treated.

43. **(Previously presented)** The method of claim 36, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

44. **(Previously presented)** The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

45. **(Previously presented)** The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is D2E7.

46. **(Canceled)**

47. **(Canceled)**

48. **(Currently amended)** A low dose method of improving symptoms in the joints of a subject having arthritis comprising administering to the subject a low dose of 0.01-0.1 mg/kg of a human anti-TNFα antibody, or antigen-binding portion thereof, ~~comprising 0.01-0.1 mg/kg~~ such that at least one symptom selected from the group consisting of inflammation, cartilage erosion, bone erosion, and vascularity is improved.

49. **(Previously presented)** The method of claim 44, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is D2E7.